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Help | Logout | Interrupt

Main Menu | Search Form | Posting Counts | Show S Numbers | Edit S Numbers | Preferences

Search Results -

Term	Documents
ICAM$	0
ICAM.DWPI,EPAB,JPAB,USPT,PGPB.	1675
ICAMA.DWPI,EPAB,JPAB,USPT,PGPB.	2
ICAMAKER.DWPI,EPAB,JPAB,USPT,PGPB.	1
ICAMA-PHOTO-EQUIP.DWPI,EPAB,JPAB,USPT,PGPB.	1
ICAMBASED.DWPI,EPAB,JPAB,USPT,PGPB.	1
ICAMBINDING.DWPI,EPAB,JPAB,USPT,PGPB.	1
ICAMCOX.DWPI,EPAB,JPAB,USPT,PGPB.	2
"ICAMCOX-OBJ2D.F2".DWPI,EPAB,JPAB,USPT,PGPB.	1
"ICAMCOX-OBJ2D.F2.SUB".DWPI,EPAB,JPAB,USPT,PGPB.	1
(ICAM$ SAME (VACCIN$)).USPT,PGPB,JPAB,EPAB,DWPI.	45

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Refine Search: `icam$ same (vaccin$)` Clear

Search History

Today's Date: 7/18/2001

DB Name	Query	Hit Count	Set Name
USPT,PGPB,JPAB,EPAB,DWPI	icam$ same (vaccin$)	45	L5
USPT,PGPB,JPAB,EPAB,DWPI	icam$ same (vaccin$ or adjuvant$) and (candida or fungus or fungal)	24	L4
USPT,PGPB,JPAB,EPAB,DWPI	icam$ same (vaccin$ or adjuvant$)	100	L3
USPT,PGPB,JPAB,EPAB,DWPI	candida same icam$	14	L2
USPT,PGPB,JPAB,EPAB,DWPI	pascual-david$	1	L1

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L4: Entry 5 of 24 File: USPT Apr 17, 2001

DOCUMENT-IDENTIFIER: US 6218166 B1
TITLE: Adjuvant incorporation into antigen carrying cells: compositions and methods

DEPR:
MPL also activates and recruits macrophages (Verma et al., 1992). Tomai and Johnson (1989) showed that MPL-stimulated T cells enhance IL-1 secretion by macrophages. MPL is also known to activate superoxide production, lysozyme activity, phagocytosis, and killing of Candida in murine peritoneal macrophages (Chen et al., 1991).

DEPR:
Based on the bladder cancer studies and the principles described in this application (see, e.g., Example 8), it is contemplated that adhesive molecules, such as fibronectin and ICAM-1, may be incorporated onto the cell surface to enable attachment of bacteria, such as BCG, on to tumor cell surfaces to improve the efficacy of the tumor cell vaccine.

DEPR:
MCV is produced in large batches and analyzed for MAA antigen expression to determine variance between lots. The MCV is screened for viral (HIV, hepatitis), bacterial and fungal infectious organisms. Equal amounts of each line are pooled to a total of 24.times.10.sup.6 cells in serum-free medium containing 10% dimethyl sulfoxide and cryopreserved in liquid nitrogen. Before cryopreservation, the cells are irradiated to 100 GY.

DEPR:
The pharmaceutical forms suitable for injectable use include sterile aqueous solutions or dispersions; formulations including sesame oil, peanut oil or aqueous propylene glycol; and sterile powders for the extemporaneous preparation of sterile injectable solutions or dispersions. In all cases the form must be sterile and must be fluid to the extent that easy syringability exists. It must be stable under the conditions of manufacture and storage and must be preserved against the contaminating action of microorganisms, such as bacteria and fungi.

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                                     bb                 ll
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222222   3333   222222
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7/18/01

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L4: Entry 6 of 24 File: USPT Oct 17, 2000

DOCUMENT-IDENTIFIER: US 6133029 A
TITLE: Replication defective viral vectors for infecting human cells

BSPR:
This aspect of the invention has a further advantage over other systems that might be expected to function in a similar manner, in that the presenter cells are fully viable and healthy, and no other viral antigens (which may well be immunodominant) are expressed. This presents a distinct advantage since the antigenic epitopes expressed can be altered by selective cloning of sub-fragments of the gene for the antigen into the recombinant retrovirus, leading to responses against immunogenic epitopes which may otherwise be overshadowed by immunodominant epitopes. Such an approach may be extended to the expression of a peptide having multiple epitopes, one or more of the epitopes being derived from different proteins. Further, this aspect of the invention allows efficient stimulation of cytotoxic T lymphocytes (CTL) directed against antigenic epitopes, and peptide fragments of antigens encoded by sub-fragments of genes, through intracellular synthesis and association of these peptide fragments with MHC Class I molecules. This approach may be utilized to map major immunodominant epitopes for CTL. In addition, the present invention provides for a more efficient presentation of antigens through the augmentation or modification of the expression of presenting accessory proteins (e.g., MHC I, ICAM-1, etc.) in antigen presenting cells. Such an approach may involve a recombinant retrovirus carrying a vector construct which directs expression of both an antigen (e.g., a tumor antigen) and an MHC protein (e.g., Class I or II) capable of presenting the antigen (or a portion thereof) effectively to T lymphocytes so that it stimulates an immune response in an animal. This offers the advantage that antigen presentation may be augmented in cells (e.g., tumor cells) which have reduced levels of MHC proteins and a reduced ability to stimulate an immune response. The approach may additionally involve a recombinant retrovirus carrying a vector construct which directs expression of both an antigen and a protein stimulating increased MHC protein expression in cells (e.g., interferon). The retrovirus infected cells may be used as an immunostimulant, immunomodulator, or vaccine, etc.

DEPR:
In a similar manner to the preceding embodiment, the retroviral vector construct can carry a gene for phosphorylation, phosphoribosylation, ribosylation, or other metabolism of a purine- or pyrimidine-based drug. This gene may have no equivalent in mammalian cells and might come from organisms such as a virus, bacterium, fungus, or protozoan. An example of this would be the E. coli guanine phosphoribosyl transferase gene product, which is lethal in the presence of thioxanthine (see Besnard et al., Mol. Cell. Biol. 7:4139-4141, 1987). Conditionally lethal gene products of this type have potential application to many presently known purine- or pyrimidine-based anticancer drugs, which often require intracellular ribosylation or phosphorylation in order to become effective cytotoxic agents. The conditionally lethal gene product could also metabolize a nontoxic drug, which is not a purine or pyrimidine analogue, to a cytotoxic form (see Searle et al., Brit. J. Cancer 53:377-384, 1986).

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222222   3333     77
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7/18/01

L4: Entry 13 of 24 File: USPT Jan 18, 2000

DOCUMENT-IDENTIFIER: US 6015694 A
TITLE: Method for stimulating an immune response utilizing recombinant alphavirus particles

BSPR:
In still another aspect, the present invention provides recombinant alphavirus particles which carry a vector capable of directing the expression of a palliative in cells infected with the alphavirus particle, the palliative being capable of inhibiting a function of a pathogenic agent necessary for pathogenicity. In various embodiments, the pathogenic agent is a virus, fungi, protozoa, or bacteria, and the inhibited function is selected from the group consisting of adsorption, replication, gene expression, assembly, and exit of the pathogenic agent from infected cells. In other embodiments, the pathogenic agent is a cancerous cell, cancer-promoting growth factor, autoimmune disorder, cardiovascular disorders such as restenosis, osteoporosis and male pattern baldness, and the inhibited function is selected from the group consisting of cell viability and cell replication. In further embodiments, the vector directs the expression of a toxic palliative in infected target cells in response to the presence in such cells of an entity associated with the pathogenic agent; preferably the palliative is capable of selectively inhibiting the expression of a pathogenic gene or inhibiting the activity of a protein produced by the pathogenic agent. In still further embodiments, the palliative comprises an inhibiting peptide specific for viral protease, an antisense RNA complementary to RNA sequences necessary for pathogenicity, a sense RNA complementary to RNA sequences necessary for pathogenicity, or a defective structural protein of a pathogenic agent, such protein being capable of inhibiting assembly of the pathogenic agent.

DEPR:
Representative examples of other pro-drugs which may be utilized within the context of the present invention include: E. coli guanine phosphoribosyl transferase which converts thioxanthine into toxic thioxanthine monophosphate (Besnard et al., Mol. Cell. Biol. 7:4139-4141, 1987); alkaline phosphatase, which will convert inactive phosphorylated compounds such as mitomycin phosphate and doxorubicin-phosphate to toxic dephosphorylated compounds; fungal (e.g., Fusarium oxysporum) or bacterial cytosine deaminase, which will convert 5-fluorocytosine to the toxic compound 5-fluorouracil (Mullen, PNAS 89:33, 1992); carboxypeptidase G2, which will cleave the glutamic acid from para-N-bis (2-chloroethyl) aminobenzoyl glutamic acid, thereby creating a toxic benzoic acid mustard; and Penicillin-V amidase, which will convert phenoxyacetabide derivatives of doxorubicin and melphalan to toxic compounds (see generally, Vrudhula et al., J. of Med. Chem. 36(7):919-923, 1993; Kern et al., Canc. Immun. Immunother. 31(4):202-206, 1990).

DEPR:
Within other aspects of the present invention, a wide variety of proteins or other cellular constituents may be carried by the alphavirus vector construct. Representative examples of such proteins include native or altered cellular components, as well as foreign proteins or cellular constituents, found in for example, viruses, bacteria, parasites or fungus.

DEPR:
Within other aspects of the present invention, alphavirus vector constructs are provided which direct the expression of immunogenic portions of antigens from foreign organisms or other pathogens. Representative examples of such antigens include bacterial antigens (e.g., E. coli, streptococcal,

staphylococcal, mycobacterial, etc.), fungal antigens, parasitic antigens, and viral antigens (e.g., influenza virus, Human Immmunodeficiency Virus ("HIV"), Hepatitis A, B and C Virus ("HAV", "HBV" and "HCV", respectively), Human Papiloma Virus ("HPV"), Epstein-Barr Virus ("EBV"), Herpes Simplex Virus ("HSV"), Hantavirus, TTLV I, HTLV II and Cytomegalovirus ("CMV"). As utilized within the context of the present invention, "immunogenic portion" refers to a portion of the respective antigen which is capable, under the appropriate conditions, of causing an immune response (i.e., cell-mediated or humoral). "Portions" may be of variable size, but are preferably at least 9 amino acids long, and may include the entire antigen. Cell-mediated immune responses may be mediated through Major Histocompatability Complex ("MHC") class I presentation, MHC Class II presentation, or both.

DEPR:
More specifically, within one aspect of the present invention, compositions and methods are provided for stimulating an immune response (either humoral or cell-mediated) to a pathogenic agent, such that the pathogenic agent is either killed or inhibited. Representative examples of pathogenic agents include bacteria, fungi, parasites, viruses and cancer cells.

DEPR:
Within one embodiment of the invention the pathogenic agent is a virus, and methods are provided for stimulating a specific immune response and inhibiting viral spread by using recombinant alphavirus viral particles designed to deliver a vector construct that directs the expression of an antigen or modified form thereof to susceptible target cells capable of either (1) initiating an immune response to the viral antigen or (2) preventing the viral spread by occupying cellular receptors required for viral interactions. Expression of the vector nucleic acid encoded protein may be transient or stable with time. Where an immune response is to be stimulated to a pathogenic antigen, the recombinant alphavirus is preferably designed to express a modified form of the antigen which will stimulate an immune response and which has reduced pathogenicity relative to the native antigen. This immune response is achieved when cells present antigens in the correct manner, i.e., in the context of the MHC class I and/or II molecules along with accessory molecules such as CD3, ICAM-1, ICAM-2, LFA-1, or analogues thereof (e.g., Altmann et al., Nature 33&512, 1989). Cells infected with alphavirus vectors are expected to do this efficiently because they closely mimic genuine viral infection and because they: (a) are able to infect non-replicating cells, (b) do not integrate into the host cell genome, (c) are not associated with any life threatening diseases, and (d) express high levels of heterologous protein. Because of these differences, alphavirus vectors can easily be thought of as safe viral vectors which can be used on healthy individuals for vaccine use.

DEPR:
In a similar manner to the preceding embodiment, the alphavirus vector construct can carry a gene for phosphorylation, phosphoribosylation, ribosylation, or other metabolism of a purine- or pyrimidine-based drug. This gene may have no equivalent in mammalian cells and might come from organisms such as a virus, bacterium, fungus, or protozoan. An example of this would be the E. coli guanine phosphoribosyl transferase gene product, which is lethal in the presence of thioxanthine (see Besnard et al., Mol. Cell. Biol. 7:4139-4141, 1987). Conditionally lethal gene products of this type (also referred to as "pro-drugs" above) have application to many presently known purine- or pyrimidine-based anticancer drugs, which often require intracellular ribosylation or phosphorylation in order to become effective cytotoxic agents. The conditionally lethal gene product could also metabolize a nontoxic drug which is not a purine or pyrimidine analogue to a cytotoxic form (see Searle et al., Brit. J. Cancer 53:377-384, 1986).

DEPR:
Specifically, within one aspect of the present invention, compositions and methods are provided for stimulating an immune response (either humoral or cell-mediated) to a pathogenic agent, such that the pathogenic agent is either killed or inhibited. Representative examples of pathogenic agents of veterinary importance include bacteria, fungi, parasites and viruses.

CLPR:
6. The method according to claim 1 wherein said antigen is obtained from a

bacteria, parasite or fungus.

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                                     bb                  ll
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7/18/01

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L4: Entry 16 of 24 File: USPT Apr 6, 1999

DOCUMENT-IDENTIFIER: US 5891841 A
TITLE: Methods of using intercellular adhesion molecule-3 (ICAM-3), antibodies thereto, and soluble fragments thereof

DEPR:
The agents of the present invention may be obtained by: natural processes (for example, by inducing an animal, plant, fungi, bacteria, etc., to produce a non-immunoglobulin antagonist of ICAM-3, or by inducing an animal to produce polyclonal antibodies capable of binding to ICAM-3); by synthetic methods (for example, by synthesizing ICAM-3, functional derivatives of ICAM-3, or protein antagonists of ICAM-3 (either immunoglobulin or non-immunoglobulin)); by hybridoma technology (for example, to produce monoclonal antibodies capable of binding to ICAM-3); or by recombinant technology (such as, for example, to produce the agents of the present invention in diverse hosts (i.e., yeast, bacteria, fungi, cultured mammalian cells, etc.), using a recombinant plasmids or viral vectors). The choice of which method to employ will depend upon factors such as convenience, desired yield, etc. However, it is not necessary to employ only one of the above-described methods, processes, or technologies to produce a particular anti-inflammatory agent; the above-described processes, methods, and technologies may be combined in order to obtain a particular agent.

DEPR:
Mice were injected with a combination of adjuvant and ICAM-3 protein which was purified from SKW3 or tonsil cells as described above. Monoclonal antibodies from immunized animals were generated using procedures known in the art.

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                                    bbb                lll
                                     bb                 ll
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22  22       44  88  88
222222     4444  8888
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7/18/01

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End of Result Set

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L4: Entry 24 of 24 File: USPT Mar 1, 1994

DOCUMENT-IDENTIFIER: US 5290551 A
TITLE: Treatment of melanoma with a vaccine comprising irradiated autologous melanoma tumor cells conjugated to a hapten

DEPR:
Recently a new preparation of IL2 has become available, which is covalently linked to polyethylene glycol (PEG). PEG-IL2 has a much longer pharmacological half-life than unmodified IL2 i.e., weekly administration results in sustained blood levels (Investigator's Brochure, Cetus Corporation). Furthermore, the toxicity of weekly administration of PEG-IL2 is mild when the weekly dose is below 1.times.10.sup.6 IU/M.sup.2. It was found that the administration of low dose IL2 to patients whose tumor have become infiltrated with activated T cells results in expansion of those cells and more potent antitumor effects. Patients with metastatic melanoma were treated using an immunotherapy regimen with the following components: 1) vaccine consisting of autologous tumor cells conjugated to DNP; 2) low dose CY pretreatment; and 3) PEG-IL2 given weekly following vaccine injection. Patients were evaluated to determine whether tumor regression had occurred, to monitor tumor inflammatory responses, and to measure DTH to autologous melanoma cells, DNFB (the form of DNP used for skin sensitization), DNP-conjugated autologous lymphocytes, diluent (Hanks solution), PPD, and recall antigens (candida, trichophyton, and mumps). Patients who are considered to be deriving benefit (clinical or immunological) from the therapy are continued in the immunotherapy regimen. Subsequent vaccines may be given without CY.

DEPR:
Fifteen patients (including 3 patients from Example 2) were treated with metastatic melanoma using a novel form of immunotherapy, i.e., tumor cell vaccine conjugated to DNP. Patients were sensitized to DNP by topical application o 5% dinitrochlorobenzene. Then every 4 weeks they received cyclophosphamide 300 mg/M.sup.2 followed 3 days later by injection of 10-25.times.10.sup.6 autologous, irradiated melanoma cells conjugated to DNP. Most patients (92%) developed delayed-type hypersensitivity (DTH) to DNP-conjugated autologous lymphocytes or tumor cells (mean DTH=17 mm). The vaccine induced a striking inflammatory response in sc and nodal metastases in 11/15 patients, consisting of erythema, swelling, warmth, and tenderness around tumor masses, and, in one case, purulent drainage. Biopsies showed infiltration with lymphocytes, which, by immunopathological and flow cytometric analyses, were mainly CD3+, CD4-, CD8+, HLA-DR+T cells. The melanoma cells in these tissues strongly expressed ICAM-1, which serves as an adhesion molecule for T cells. Thus, DNP-vaccine seems to induce a degree of anti-melanoma immunity not seen with previously tested immunotherapy.

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7/18/01

WEST

Generate Collection

L2: Entry 3 of 14 File: USPT Sep 28, 1999

DOCUMENT-IDENTIFIER: US 5958406 A
TITLE: Acne treatment with multifunctional enzyme

BSPR:
The invention further provides (a) methods for treating or prophylactically preventing a cell-cell or cell-virus adhesion syndrome comprising administering an anti-adhesion effective amount of a hydrolase effective to remove or inactivate a cellular or viral acceptor or receptor adhesion component that is involved in the cell-cell or cell-virus adhesion, (b) compositions or substances for use in such methods, (c) pharmaceutical compositions containing effective amounts of enzyme for use in such methods, and (d) uses of the enzyme composition for manufacturing a medicament for use in such methods. Preferably, the syndrome comprises inflammation, shock, tumor metastases, autoimmune disease, transplantation rejection reactions or microbial infections. Preferably, (a) the syndrome is selected from the group consisting of microbial infection, immune disorder, cystic fibrosis, COPD, atherosclerosis, cancer, asthma, septic shock, toxic shock syndrome, conjunctivitis, reperfusion injury and pain, and (b) a cell surface receptor, associated with the cell-cell or cell-virus adhesion syndrome, selected from the group consisting of ICAM-1, ICAM-2, VCAM-1, CD4, CD8, CD11, CD18, CD28, CD29D, CD31, CD44, CD 49, CD62L, CD102 and asialo GM1 ceramide is removed or inactivated by the administered hydrolase. Preferably, a microbial infection is treated or prevented and the microbial infection is a herpes, HIV, hepatitis or papilloma infection; an infection causing colitis, ulcer or diarrhoea; a candida infection, such as an oral, vaginal or esophageal candida infection; a cold or influenza infection; a pseudomonas, haemophilus, staphylococcus, streptococcus, klebsiella or E. coli infection; a primary or secondary infection of leprosy; or an infection causing conjunctivitis.